

Refining the diagnosis of complete and partial moles: Role of p57 immunostaining in a tertiary care setting

Umber Adnan¹, Rabia Ahmed², Akhter Ali Bajwa², Sadia Talib³, Naveed Khan⁴, Vareesha Haider²

¹Karachi Institute of Medical Sciences, Malir Karachi Pakistan / NUST School of Health Sciences, Islamabad Pakistan

²PNS Shifa Hospital, Karachi Pakistan

³Karachi Institute of Medical Sciences, Malir Karachi Pakistan

⁴Combined Military Hospital (Karachi Institute of Medical Sciences), Malir Karachi Pakistan

ABSTRACT

Objective: To assess the utility of p57 immunohistochemistry to enhance the diagnostic accuracy of histopathology in differentiating complete and partial hydatidiform moles.

Material and Methods: An ambispective cross-sectional diagnostic utility study was conducted at the PNS Shifa Hospital, Karachi, and the Karachi Institute of Medical Sciences. A total of 54 hydatidiform mole cases collected between 1st April 2021 and 30th March 2026 were included. Initial diagnoses were based on histomorphological assessment of Hematoxylin and eosin (H&E)-stained sections, followed by p57 immunohistochemistry (IHC) for final classification as complete or partial hydatidiform mole. Associations between histomorphological features and mole type were analyzed using Chi-square test or Fisher's exact test. Agreement between initial histomorphological and final p57-based diagnoses was assessed using Cohen's kappa coefficient. Statistical analysis was performed using SPSS version 26.

Results: Histomorphological assessment demonstrated significant differences between complete and partial hydatidiform moles across the evaluated morphological features ($p < 0.05$). Initially 35(64.8%) cases were classified as complete hydatidiform mole (CHM), and 19(35.1%) as partial hydatidiform mole (PHM). There was substantial agreement between the initial histomorphological and final p57-based diagnoses ($\kappa = 0.750$, $p < 0.001$). Following p57 immunohistochemistry, six discordant cases were reclassified, yielding final diagnoses of 37(68.5%) CHMs and 17 (31.5%) PHMs.

Conclusion: p57 immunohistochemistry provides a reliable and practical adjunct to histomorphology, improving diagnostic accuracy and supporting appropriate clinical management of hydatidiform moles.

Keywords: Complete hydatidiform mole, Hydatidiform mole, Immunohistochemistry, Partial hydatidiform mole, p57 Protein.

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INTRODUCTION

Hydatidiform mole (HM) is a type of Gestational Trophoblastic Disease (GTD) arising from the placenta and characterized by abnormal trophoblastic proliferation. Histologically they are considered benign, pre-malignant lesions. HMs are classified as Complete moles (CHMs) and partial moles (PHMs) [1,2]. 80-90% of CHMs result when an empty oocyte is fertilized by a single sperm that later duplicates its genome (monospermic homozygous), while 10-20% result due to fertilization of an empty oocyte by two

sperms (dispermic heterozygous). On the other hand, PHMs are triploid conceptions, with >95% cases resulting from fertilization of an ovum by two sperms (dispermic heterozygous). Very few of the cases are monospermic homozygous, due to fertilization of an egg with a sperm, followed by duplication of the paternal genome [3].

The global incidence of HM is 1-2 per 1000 pregnancies, and varies across the regions. Considerably higher rates have been documented from South-east Asia (3.8-13/1000 pregnancies), whereas western Europe and North America reported the lowest rates (0.8-1.5) [5,6].

Comprehensive national data regarding GTD epidemiology is deficient in Pakistan, and there are no population-based studies on true incidence of HM. Recent hospital-based studies continue to highlight regional variations in disease burden, with a reported frequency of 3.72 cases per 1000 pregnancies in a tertiary care setting [7,8].

Correspondence: Dr. UMBER ADNAN, Assistant Professor, Department of Pathology, NUST School of Health Sciences, Islamabad Pakistan

Email: umberadnan08@gmail.com

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Classification of HM into CHM and PHM is clinically very significant for treatment and prognosis, as the former has an increased risk to progress to persistent GTD or gestational trophoblastic neoplasm (GTN) [9]. This categorization can be achieved through histomorphological evaluation, immunohistochemical (IHC) studies, or genetic testing which is the gold standard. Although CHMs and PHMs have distinct morphological features, overlap in histopathological findings makes this distinction challenging. In addition, subjective interpretation of diagnostic criteria often leads to interobserver and intra-observer variability [10, 11].

This challenge is further accentuated in early gestation molar specimens, as characteristic histopathological features are often insufficiently developed or absent. Among these, very early complete moles (VECMs) are especially important to recognize accurately because of their higher malignant potential [9, 10, 11].

A Cyclin dependent kinase inhibitor protein, p57, has proven to be an adjunct IHC marker to solve this issue. P57 is encoded by a gene that has its paternal copy imprinted (silenced), and maternal copy expressed (active). This unique expression is exploited to differentiate PHMs from CHMs – that lack maternal genetic material and hence do not express p57 [2,11].

In low-income countries like Pakistan, the high cost, lack of infrastructure, and limited technical expertise restrict the use of genetic testing for diagnosing HMs in routine clinical practice. National data on the usefulness of p57 immunomarker in accurate diagnosis of HMs remains deficient. There is a dire need to generate local evidence in this regard as the incidence of HMs in our population is considerably high. The present study was conducted to evaluate the utility of p57 as an adjunct tool for differentiating partial from complete moles in our population, as accurate classification is critical for risk stratification, appropriate follow-up and clinical management.

MATERIAL AND METHODS

This was an ambispective cross sectional diagnostic utility study, incorporating both retrospective and prospective components, conducted from 1st April 2021 to 30th March 2026 at the Department of Histopathology, PNS Shifa hospital, Karachi, in collaboration with Karachi Institute of Medical Sciences. Ethical approval was obtained from the Institutional Review Board (vide letter number ERC/HST/100) prior to commencement of the study. The requirement for informed consent was waived for

archived retrospective cases retrieved from departmental records. Written informed consents were obtained from all prospectively enrolled cases.

A total of 54 hydatidiform mole cases were included, comprising 30 archived formalin-fixed paraffin-embedded (FFPE) cases diagnosed by histomorphological assessment between April 2021 and December 2023, retrieved for p57 IHC testing, and 24 consecutive cases prospectively enrolled from January 2024 to March 2026 that underwent preliminary histomorphological assessment followed by p57 IHC for final diagnosis.

Products of non-molar gestations, hydropic abortuses, and invasive moles were excluded.

The sample size was estimated using Cochran's formula for proportions assuming a 95% confidence level ($Z = 1.96$) and a margin of error of 5% ($e = 0.05$). In the absence of definitive local prevalence data, the expected proportion (p) was set at 0.5 to yield the maximum required sample size, as recommended in standard epidemiological methodologies. Applying the Finite Population Correction for a single-center design, the adjusted minimum required target was 48 cases. The calculations were verified using the OpenEpi sample size calculator. Accordingly, all eligible archived and prospectively enrolled cases were included, yielding a final sample size of 54 cases [12,13].

Pertinent information and demographic details were recorded on standardized case record forms.

For histological examination, 3–5 μ m thick sections were cut from formalin- fixed paraffin-embedded blocks and stained with hematoxylin and eosin (H&E). Initial diagnoses were made by experienced histopathologists based on histomorphological features alone, and cases were classified as CHM or PHM.

For evaluation of p57 immunohistochemistry, 3–5 μ m thick sections were mounted on adhesive coated glass slides. After heat induced epitope retrieval, they were subjected to immunohistochemistry by Streptavidin-biotin peroxidase complex technique using Quartett p57KIP2 mouse monoclonal antibody. The immunostaining results were interpreted based on presence or absence of nuclear staining in the cytotrophoblasts, and villous stromal cells. The p57 stain was considered “negative” if the cytotrophoblasts and villous stromal cells were either entirely negative or showed nuclear staining in <10% of cells. The p57 staining was considered “positive” if either both these cells expressed distinct diffuse nuclear staining ($\geq 50\%$) or focal non-diffuse staining ($\geq 10\%$ but <50%). The nuclear expression of

intermediate trophoblasts and decidual cells served as internal positive controls. "Negative" results were interpreted as consistent with CHM, whereas "Positive" results supported the diagnosis of PHM. Subsequently, revised final diagnoses were made by the histopathologists following p57 immunostaining as a part of structured diagnostic assessment. All the results were systematically documented for analysis. The statistical analysis was performed using SPSS version 26. Age was described as mean and standard deviation (SD), and for analytical purposes was categorized into predefined groups. Categorical variables including villous enlargement and edema, cistern formation, villous contour and shape, trophoblastic proliferation, cytologic atypia, presence of fetal vessels/ nucleated red blood cells (RBCs), initial histomorphological diagnoses, and final immunohistochemistry-based diagnoses were expressed as frequencies and percentages [n (%)]. Associations between categorical variables, such as histomorphological features and type of HMs were assessed using Chi-square test or Fisher's exact test where appropriate. A p value <0.05 was considered significant. Agreement between the initial histomorphological and final p57-based diagnoses was assessed using Cohen's kappa coefficient.

RESULTS

Out of the 54 patients included in the study, majority, 32 (59.3%) belonged to the 21-30 years age group. Lesser number of cases, 16 (29.6%) were 31-40 years of age, and only few cases were seen at the extremes of age with 7.4% cases being ≥ 40 years and only 3.7% were ≤ 20 years old, as shown in Table-I.

The histomorphological features considered during the initial classification of cases as Complete or Partial Hydatidiform moles are summarized in Table-II, and examples are shown in Figures-I and II. Diffuse villous enlargement and edema, was appreciated in 30 (85.7%) cases of CHM, compared to only 02 (10.5%) of PHM cases. Whereas, most of

the PHM cases 17 (89.4%) showed focal villous enlargement (p value <0.001). Prominent, large cisterns were predominantly seen in 24 (68.6%) of CHMs, and only 02 (10.5%) of PHMs, (p value <0.001). The villous contour was smooth in majority 23 (65.7%) of CHMs, while it was scalloped irregular in most 13 (68.4%) of PHMs, (p=0.013). Circumferential, moderate to marked trophoblastic proliferation was observed in 22 (77.1%) of CHMs compared to 02 (10.5%) of PHMs (p value <0.001). The trophoblastic proliferation was predominantly focal, and mild, in 17 (89.5%) of PHMs. Moderate to marked cytologic atypia was seen in 21 (60%) of CHM cases compared to 04 (21.1%) of PHM cases (p= 0.006). Nucleated red blood cells were present in 13 (68.4%) of PHMs, while they were absent in all cases of CHM (p = 0.001).

A subset of complete mole cases showed features consistent with VECMs, characterized by subtle villous enlargement, irregular villous contours, and focal trophoblastic hyperplasia. Representative photomicrographs are shown in Figure-II (e, f).

Based on histomorphological features, preliminary diagnoses of 35 (64.8%) CHMs and 19 (35.1%) PHMs were made, as shown in Table-III. After p57 immunostaining analysis, refined, final diagnoses were established. Of the 35 (64.8%) histomorphologically diagnosed CHM cases, 33 (94.2%) expressed negative, and 02 (5.7%) showed positive nuclear staining. Whereas, among the 19 (35.1%) histomorphologically classified cases of PHM, 15 (78.9%) showed positive, and 04 (21.0%) showed negative nuclear staining. Overall diagnostic concordance between H&E morphology and p57 expression was observed in 48/54 (88.9%), while diagnostic discordance and subsequent reclassification occurred in 6/54 (11.1%) of cases.

Comparison of the initial histomorphological diagnosis with the final p57-based diagnosis demonstrated an overall concordance in 48/54 (88.9%) cases. Agreement between the two methods was substantial (Cohen's $\kappa = 0.750$, p<0.001).

Table-I: Age distribution of patients (n= 54).

Age Category	Number of Patients
≤ 20	02 (3.7)
21-30	32 (59.3)
31-40	16 (29.6)
≥ 40	04 (7.4)
Total	54 (100)

Mean age: 29.63 years (SD 6.15)

Table II: Comparison of Histomorphological features in Complete and Partial Moles

Histomorphological Feature	Category	Complete Mole n (%)	Partial Mole n (%)	P value
Villous enlargement and edema	Diffuse	30 (85.7%)	02 (10.5%)	< 0.001
	Focal	05 (14.3%)	17 (89.4%)	
Cisterns	Absent	0	02 (10.5%)	

	Present, smaller, less frequent	11 (31.4%)	16 (84.2%)	< 0.001*
Villous contour and shape	Prominent, large	24 (68.6%)	01(5.2%)	0.013
	Smooth	23 (65.7%)	06 (31.6%)	
	Scalloped, irregular	12 (34.3%)	13 (68.4%)	
Trophoblastic proliferation	Focal, mild	08 (22.9%)	17 (89.5%)	< 0.001
	Circumferential, moderate to marked	27 (77.1%)	02 (10.5%)	
Cytologic atypia	Mild	14 (40%)	15 (78.9%)	0.006
	Moderate to marked	21 (60%)	04 (21.1%)	
Fetal vessels/ nucleated RBCs	Absent	35 (100%)	13 (68.4%)	0.001*
	Present	0	06 (31.6%)	

*Fisher's Exact test applied where appropriate

Table-III: Correlation of Initial histomorphological diagnosis with final p57 immunohistochemistry-based diagnosis (n=54).

Initial Histomorphological Diagnosis	Total Cases n (%)	Final P57 IHC Based Diagnosis	
		P57 Negative (CHM)	p57 Positive (PHM)
Complete Hydatidiform Mole (CHM)	35 (64.8%)	33 (94.3%) (Concordant)	02 (5.7%) (Discordant)
Partial Hydatidiform Mole (PHM)	19 (35.2%)	04 (21.1%) (Discordant)	15 (78.9%) (Concordant)
Total	54 (100%)	37 (68.5%)	17(31.5%)

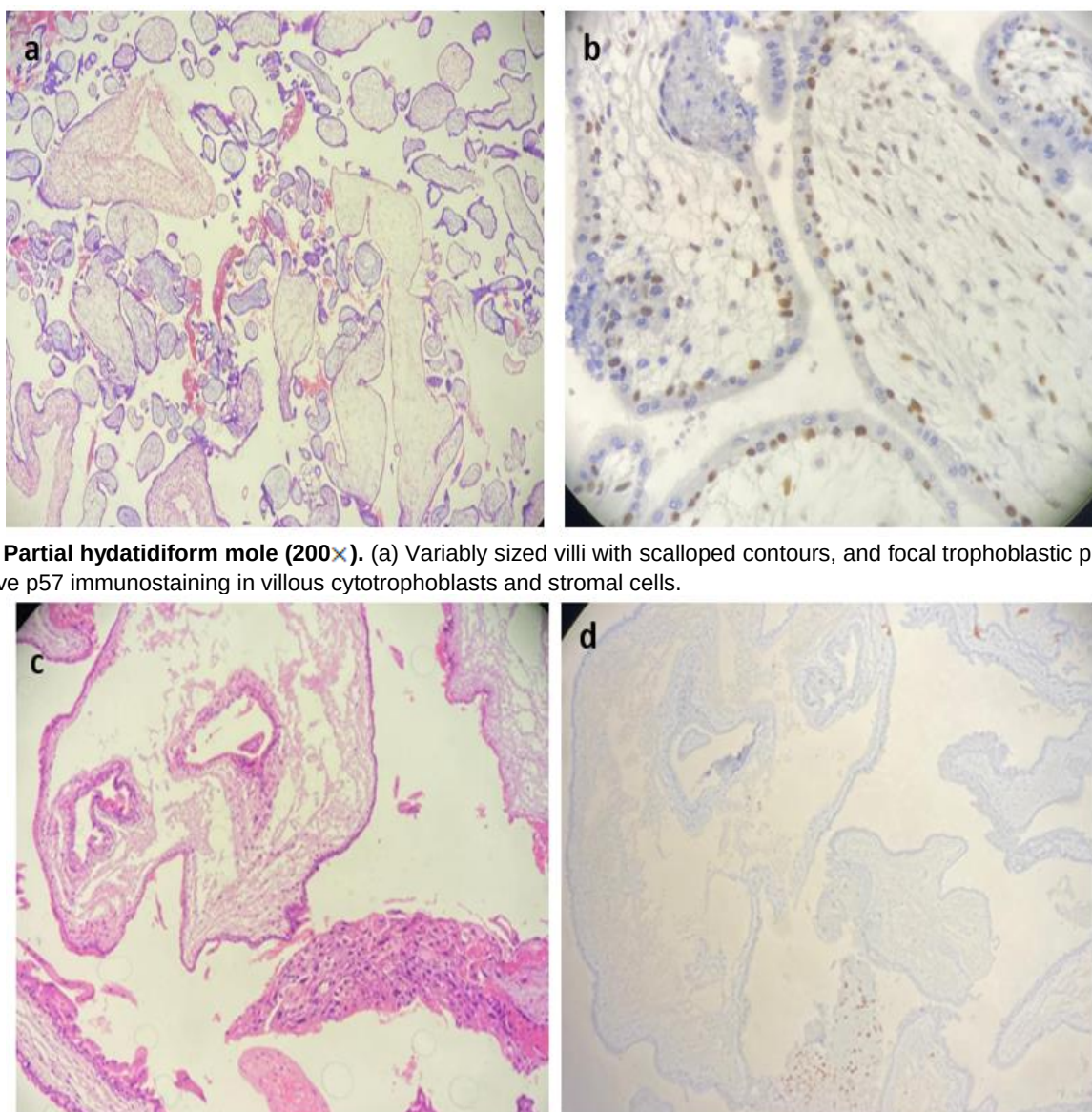


Figure-I: Partial hydatidiform mole (200x). (a) Variably sized villi with scalloped contours, and focal trophoblastic proliferation. (b) Positive p57 immunostaining in villous cytotrophoblasts and stromal cells.

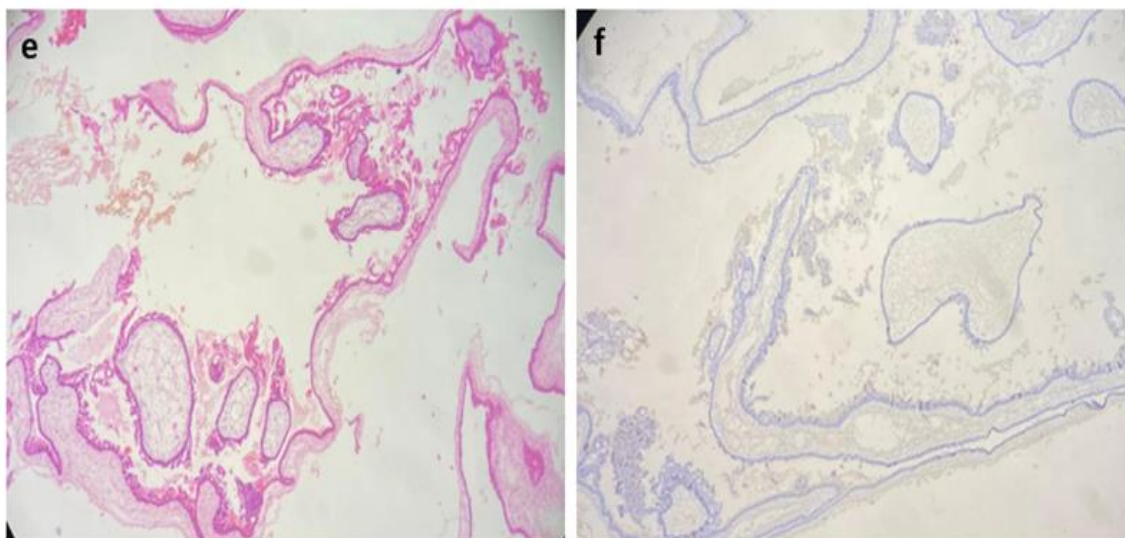


Figure-II: Complete hydatidiform mole (c, d) and very early complete hydatidiform mole (e, f) (200×). (c) Hydropic villi with circumferential trophoblastic hyperplasia, and cisterns. (d) Negative p57 immunostaining in villous cytotrophoblasts and stromal cells, with positive intermediate trophoblasts as an internal control. (e) Bulbous villous enlargement with trophoblastic hyperplasia and myxoid stroma. (f) Lack of p57 expression in villous cytotrophoblasts and stromal cells, with preserved positive internal control.

DISCUSSION

In the present study, patients predominantly belonged to the 21-30 years age bracket. This can be attributed to the increased number of molar pregnancies during the peak reproductive age. While extremes of age, like adolescence and age above 35 years, are documented risk factors for molar gestation [10,14], our findings support the national and international data reporting an increased incidence of HMs in the same age group [9,15]. Genetic and environmental factors, as well as ethnicity, might be responsible for the variation in demographic patterns.

The diagnosis of HM relies on characteristic histomorphological features that distinguish CHM from PHM [16]. Most of our CHM cases were classically associated with diffuse villous enlargement and edema, prominent cisterns, and circumferential trophoblastic proliferation with cytologic atypia, as established in the literature and endorsed by the WHO 2020 classification. In contrast, our PHM cases frequently displayed focal villous enlargement, less frequent cisterns, focal trophoblastic proliferation, and retained fetal elements, reinforcing established diagnostic distinctions [2,6,11].

In our study, some cases lacked the classic features of CHM or PHM and displayed overlapping histomorphological characteristics; therefore, they were initially classified according to the predominant features. Some CHMs exhibited features typically associated with PHM, while some PHMs showed features favoring CHM. These overlaps highlight the

limitations of morphology alone and underscore the value of p57 IHC in diagnostically challenging cases [2,9,17].

Several factors contribute to the limited accuracy of histomorphological assessment. Significant inter- and intra-observer variability, even among experienced pathologists, may lead to misclassification in about 20% of cases [18,19]. In addition, the increasing evaluation of very early molar tissues, in which incomplete villous development often obscures the classic morphological features, making accurate classification more challenging [11,20,21].

In routine clinical practice, differentiating CHM from PHM is essential, as it guides follow-up strategy and risk assessment for GTN, which is higher for CHM (9-20%) and intermediate for PHM (0-4%) [18,22]. Previous studies have established the essential role of novel ancillary techniques, particularly p57 IHC, that demonstrates a very high concordance with genotyping, the gold standard for the classification of HM, highlighting its diagnostic reliability [10,22]. Furthermore, the simplicity, cost-effectiveness, and rapid turnaround time make this technique a highly practical diagnostic tool [22,23].

The present study emphasizes the important role of p57 IHC in the accurate classification of hydatidiform moles. Although substantial agreement was observed between the initial histomorphological and final p57-based diagnoses (Cohen's $\kappa = 0.750$, $p < 0.001$), p57 IHC led to reclassification of 6/54 (11.1%) cases. Similar findings have been reported by various studies demonstrating the value of p57 IHC in

refining histomorphology-based diagnosis [18,20,24]. In our study, p57 IHC was particularly useful in diagnosing VECMs because of their subtle and overlapping histopathological features, as reported earlier [10,23]. This likely explains the reason why four of our initial PHM cases were reclassified as CHM following p57 IHC testing. VECMs often lack morphologic features seen in more advanced lesions and may closely resemble partial moles on routine H&E examination. Conversely, marked hydropic change or sectioning artifacts in PHMs may mimic complete mole morphology, accounting for two cases reclassified from CHM to PHM.

Our findings highlight the limitations of relying solely on histomorphology, particularly in diagnostically challenging cases. Incorporating p57 immunostaining alongside routine histopathological assessment improves diagnostic accuracy. Although molecular genotyping remains the gold standard, its limited availability in resource-limited settings such as Pakistan underscores the practical value of p57 IHC as an effective adjunct diagnostic tool to support appropriate clinical management.

CONCLUSION

The addition of p57 immunohistochemistry to routine histomorphological assessment improves diagnostic accuracy and reduces misclassification of hydatidiform moles, particularly in challenging cases, with important implications for risk stratification and patient management.

LIMITATIONS / RECOMMENDATIONS FOR FUTURE RESEARCH

The relatively sample size and single-center design may limit the generalizability of the findings. Molecular genotyping was not performed; therefore, comparison with the gold standard was not possible. Larger, multi-center studies, incorporating molecular analysis are needed in the future to validate and strengthen these findings.

CONFLICT OF INTEREST

None

GRANT SUPPORT & FINANCIAL DISCLOSURE

Declared none

AUTHORS CONTRIBUTION

Umbur Adnan: Substantial contribution to study concept, acquisition of data, manuscript writing, final approval, responsible for every aspect of the research

Rabia Ahmed: Substantial contribution to conceptualization, study design, acquisition of data, interpretation of data, Critical revision of the manuscript, responsible for every aspect of the research

Akhter Ali Bajwa: Substantial contribution to study supervision, critical review, final approval, responsible for every aspect of the research

Sadia Talib: Substantial contribution to analysis of data, manuscript writing, responsible for every aspect of the research

Naveed Khan: Data acquisition, responsible for every aspect of the research

Vareesha Haider: recording of data, critical review, responsible for every aspect of the research

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